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ABSTRACT

This paper provides a critical review of the relapse process as traditionally defined within the medical or "disease" model of alcoholism. A detailed case study of a relapse is presented, as seen through the eyes of one alcoholic client. The sequence of events in this case study is used to illustrate the theoretical and experimental literature reviewed in later sections of the paper. Highlights of the medical model of relapse are then presented, with particular attention devoted to the key concepts of craving and loss of control. Following a critical review of this traditional approach, an alternative explanation of the relapse process is offered, drawing upon social learning theory and recent behavioral investigations of alcoholism and social drinking. Finally, the implications of this alternative analysis for the prevention and treatment of the alcoholic relapse are discussed. (Author)

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CRAVING FOR ALCOHOL, LOSS OF CONTROL, AND RELAPSE: A COGNITIVE-BEHAVIORAL ANALYSIS

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Abstract

Craving for alcohol and loss of control drinking are considered to be important determinants of relapse within the traditional disease model of alcoholism. The theoretical literature describing the constructs of craving and loss of control is critically reviewed, and an alternative explanation of the relapse process is offered. A cognitive-behavioral model is proposed, with an emphasis on the following determinants of relapse: the influence of high-risk situations, available alternative coping behaviors, expectations about alcohol and its effects, and the individual's cognitive reaction to taking the first drink (the Abstinence Violation Effect). Treatment implications of this model are reviewed, including skill training as a method of coping with specific high-risk situations, global alternatives to drinking, and the programmed relapse. The theory is illustrated throughout with a detailed case-study of one alcoholic's relapse.

A version of this paper will be presented at the forthcoming NATO International Conference on Behavioral Approaches to Alcoholism, to be held in Bergen, Norway, August 28 - September 1, 1977.

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The purpose of this paper is to provide a critical review of the relapse process as traditionally defined within the medical or "disease" model of alcoholism.[1] In the traditional approach, alcoholism is viewed as an addiction, and relapse is defined as the sequence of events leading to readdiction following a period of abstinence from alcohol use. This is the common usage of the term in medical parlance, and is reflected in the following definition of relapse taken from Webster's New Collegiate Dictionary: "a recurrence of symptoms of a disease after a period of improvement." This same dictionary defines addiction as "compulsive physiological need for a habit-forming drug." Consistent with this emphasis on addiction as a physiological need, proponents of the medical model frequently attribute an alcoholic's relapse to internal symptoms such as physical craving for alcohol or an involuntary, compulsive loss of control over drinking. Falling off the wagon after a period of abstinence is thus taken as a pathognomonic symptom of alcoholism.

By its very nature, the designation of alcoholism as

a disease downplays the importance of environmental and psychological factors. Many questions go unanswered because they are rarely even asked by those who subscribe to the disease model, with its exclusive emphasis on internal, physiological determinants. Are there specific environmental events which serve as triggers for relapse? Do the alcoholic's emotions, moods and feelings affect the probability that he or she will resume drinking following a period of sobriety? Are the determinants of the first drink the same as those assumed to govern subsequent drinking in the relapse process? How does the alcoholic react to and conceptualize the events leading to and following a relapse, and how do these reactions affect the individual's later drinking behavior? Is it possible to prepare alcoholics in treatment to anticipate the likelihood of a relapse, so that they may engage in preventive alternative behaviors? To borrow a term from the medical model, can we develop treatment procedures which would "inoculate" the alcoholic against the inevitability of relapse? And the most controversial question of all: can we teach alcoholics to control or moderate their drinking, thus preventing the readdiction usually associated with relapse?

I hope to provide answers to some of these questions and to raise other related issues throughout this chapter.

The plan of the chapter is as follows. I will first present a case study of a relapse seen through the eyes of one of our alcoholic clients. The sequence of events in this case study will serve as a convenient example to illustrate the theoretical and experimental literature to be reviewed in later sections. Highlights of the medical model of relapse will then be presented, with particular attention devoted to the key concepts of craving and loss of control. Following a critical review of this traditional approach, an alternative explanation of the relapse process will be offered, drawing upon social learning theory and recent behavioral investigations of alcoholism and social drinking. Finally, the implications of this alternative analysis for the prevention and treatment of the alcoholic relapse will be discussed. Until a better word becomes available, I shall use the term "relapse" to refer to the resumption of any drinking behavior (including a single drink or "slip") following a period of voluntary abstinence.

The Alcoholic Relapse: A Case Study

The following case study concerns a young woman whom we shall call Liane.[2] Liane was a recent client in our addictive behaviors treatment program at the Center for Psychological Services at the University of Washington. She first came to my attention following a lecture I had

given on alcoholism. During our initial meeting, Liane admitted that she was concerned about her own drinking, and asked to be evaluated as a possible candidate for our treatment program. A thirty-year old single woman, who lived in a self-contained apartment in her parent's home in Seattle, Liane was a senior undergraduate student majoring in psychology at the university. In the months prior to our first meeting, Liane's drinking began to interfere with her studies, and she was doing poorly in her exams. Rather than devoting time to her assignments, Liane would spend most of her evenings alone, sipping vodka and watching television in her apartment. She did not have a steady boyfriend, and reported that most of her time was spent either alone or with her parents. She claimed that she could not relate comfortably to most people unless she had been drinking. A two-week daily self-monitoring report of her alcohol intake revealed that she was consuming almost a full quart of vodka each day (drinking mostly at night), along with a variety of tranquilizers and barbituates. Her rate of drinking had steadily increased over the past several years, and it was clear that most professionals would diagnose her as alcoholic.

When I confronted her with the seriousness of her condition, she agreed to undergo a period of voluntary

hospitalization to provide medical supervision for her detoxification from both alcohol and the other drugs she had been taking. Before she was released from the hospital, she committed herself to complete abstinence from alcohol for a period of at least one year. We agreed to provide a program of supportive therapy for her during this year. All went well during the first weeks of the program, and she maintained total abstinence during this initial period. On the 58th day following her discharge from the hospital, however, Liane experienced a "slip" and consumed one mixed drink during a luncheon date with a female friend. Although she did not take another drink on that day, and maintained abstinence again for the next twenty days, she consumed an additional single drink, again during a luncheon date with the same friend, on the 78th day. Finally, on the 81st day following her discharge, Liane drank to the point of intoxication during a weekend evening alone in her apartment. The course of her drinking during the first 150 days after her hospitalization is displayed in Figure 1. An examination of this figure reveals the overall pattern of a typical relapse. Following her initial intoxication experience on the 81st day, Liane again refrained from any drinking for about two weeks, followed by another occasion of "social drinking" (limiting herself to no more than two drinks), and shortly

thereafter, another bout of intoxication. With the start of university classes in the fall, her drinking began to increase until it almost equalled her pretreatment rate of consumption. At the time of this writing, Liane has shown considerable improvement and is continuing treatment in our program.

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Insert Figure 1 About Here

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For the present purpose, I would like to examine the events associated with her first two drinks (the "slips" on the 58th and 78th days), and her first intoxication (81st day). When these events occurred, I asked Liane to provide a detailed written account of everything she could remember: the events preceding the drinking, her feelings at the time, her reactions, and any other important details which stood out in her mind. As these reports were written within a few days of the actual events, I feel reasonably confident about their accuracy. The following excerpts are taken directly from her written account. The luncheon date reported for July 17 was the first social event of any importance Liane had participated in since her release from the hospital. Sharon, Liane's luncheon companion, was a woman who lived in the same neighborhood, although they were not close friends at the time.

Thursday, July 17, 58th day: The First Drink

Lunch with neighbor Sharon. Sharon's husband was in Detroit the week of July 17th because of the death of his nephew. I know Sharon and her husband casually. When I asked her to lunch, I told her that I thought it would be nice to wander through the shops in Edmonds, and then have lunch. I had no intention of having a drink, nor did I think about how I'd handle the problem, particularly since it was I who was paying for the lunch. This was, however, the first time I had invited anyone to dine with me as my guest since I had gotten out of the hospital, and more importantly, Sharon knew nothing about my problem drinking. I picked her up at 10:30 a.m., and we went to Edmonds. Both of us were dressed up, and I felt very at ease, cheerful and confident. I had tentatively planned to eat in Edmonds, but Sharon suggested that she enjoyed eating at a restaurant in Aurora Village, so I agreed that we go there. Throughout the entire time we were together, there was pleasant and interesting conversation. When we were seated, the waitress came by and asked if we wanted cocktails. Since I was taking her to lunch, I said, "Sharon, would you like a cocktail?" She ordered a scotch and soda, and then the waitress looked at me and said, "And you?" "Gin and tonic, please," just came

out. I didn't plan it and I wasn't prepared for it.

The first sip was so strong, it literally shocked me. It was difficult for me to finish the drink. I am not entirely sure whether or not I imagined some of the physical effects, perhaps because of guilt, or if it was real. Sharon finished her drink before the lunch even came. I was just finishing when we were ready to leave, which was approximately 45 minutes from start to finish. I got a headache, my nose got stuffy, my thoughts were tangled and my voice and actions were abrasive.... There were several things that came from this that did help me. One thing was that committing myself to one year of abstinence from alcohol, after an inebriated state of some duration, was a difficult reality to keep after a month or so because there is no reality in inebriation. Therefore, I was especially vulnerable to the unreality of alcoholic romanticism used in advertising, television, and movies. This sounds trivial, but this romanticism is a stimulus for envy and anxiety in me. Alcohol consumption is so intermeshed in the structure of society that it is very difficult not to feel the influence. For me, what was going through my mind was that a social drink at parties, lunches, dinners, etc., is condoned and exploited,

making me feel extremely self-conscious for not participating. But, when one gets into the predicament of drug and alcohol abuse as myself, society doesn't fail to ostracize and condemn.

Twenty days later, on August 6, Liane again invited Sharon out for lunch, and once again she consumed a single mixed drink. Unlike the first occasion, however, Liane considered the possibility of having a drink in advance of the luncheon. The experience of July 17 had brought about a shift in her attitudes toward drinking, as she observes in the following passage:

> I can't deny that the first situation was spontaneously reinforcing, greater than I had realized. I would only be kidding myself if I were to say that repeating the same scene, i.e., having one drink at lunch, was not planned; at least I had no real intentions of not having that drink.... Having this drink was more of a pain than a pleasure. I didn't feel good about having that drink. It lowered my self-esteem and did create some guilty and remorseful feelings.

Three days later, Liane consumed enough alcohol to become intoxicated. It was a Saturday, and she had no plans for the evening. Her parents were going out that night.

Saturday, August 9, 81st day: The First Intoxication.

> I got up early, and I felt content and at ease. I

went down to the den and played the organ for several hours. I then put some records on which I hadn't done since I came home from the hospital. Unfortunately, as I recollect now, the records started my change in mood. It never occurred to me that the music conditioned specific moods in me without my being aware of it. So, as I played some records and sang along, my mood became melancholy and I began to feel sorry for myself and ill at ease. The point at which I noticed my "free floating anxiety" came around 4:00 in the afternoon. I have my little fantasies. This is not easy to write, and I don't know how to write it without sounding like I belong in a mental institution. My fantasy is to take a real person (male) whom I don't know personally, but through TV, etc., and, well, to imagine that we are together. So, suddenly, about 4:00, his face just popped into my mind. This really affected my moods, because, well, I was alone in the house, it was Saturday, and I knew I was going to be alone for the evening. I had no plans for doing anything that night, and I began to feel sad about being alone. After a while, I began to feel more and more anxious about this. At first, I thought I would feel better if I could take a tranquilizer, so I searched all over the house for the pills my mother had hidden. I couldn't find them,

which made me all the more upset. What I did find, though, was two bottles of airline scotch, miniature bottles like you buy on the plane, hidden in a teapot in the kitchen. I also found three bottles of beer in the fridge upstairs. At first, I tried to ignore them, but I couldn't. Finally, I took out the two bottles of scotch and put them on the table. I just looked at them for a long time, without even touching them. After about half an hour, I finally opened one of the bottles and poured it into a glass with some ice. I sipped the drink but didn't feel anything at first, so I poured in the other one and drank that too. After that, I drank the bottles of beer in the fridge and then I don't remember what happened. I woke up later that night with a hangover, and I felt terrible about what I had done.

I would like to discuss the implications of this case in terms of several different theoretical models of the relapse process. To begin, let us examine the major assumptions of the traditional medical approach.

Craving and Loss of Control as Determinants of Relapse: The Medical Model

In 1954, the World Health Organization sponsored a joint meeting of their Expert Committees on Mental Health

and on Alcohol to discuss the physical basis of dependence on alcohol. Craving for alcohol was one of the main topics of discussion. In one of the position papers presented at this meeting, Isbell (1955) distinguished between two kinds of craving:

> Two kinds of "craving" are postulated. The first is a "physical" or nonsymbolic craving which occurs in persons who have been drinking excessive amounts of alcohol for long periods of time, and is manifested by symptoms on withdrawal of alcohol. This type of craving is believed to be due to physiological alterations, the mechanism of which is not yet understood.... The second kind (or kinds) of craving is thought to account for initial abuse of alcohol, and for relapse after abstinence. It is postulated that this second kind of craving is chiefly psychological in origin. (Isbell, 1955, p. 42; italics added.)

In another paper in the W.H.O. report, Mardones (1955) defines craving for alcohol as "an urgent and overpowering desire to drink alcoholic beverages.... The urgency of the desire is a passive condition that is perceived by the individual, and 'overpowering' means inducing an active attitude directed to surmount the obstacles opposing the desire" (pp. 51-52).

As Isbell noted, physical craving is most often

defined as the desire for alcohol experienced by a drinker undergoing the symptoms of withdrawal following a prolonged drinking bout. The assumption here is that drinking alcohol will relieve the unpleasant and often painful physical sensations brought about by withdrawal: drinking "the hair of the dog which bit me," the saying goes. Physical craving defined in this manner resembles the notion of craving for heroin experienced by the addict undergoing withdrawal.

Many authorities, however, have extended the concept of craving to account for the circumstances leading to relapse in the abstinent alcoholic, as in Isbell's second type of craving described above. Relapse is thus precipitated by a subjectively experienced intense desire or need for alcohol, which somehow "overpowers" the volitional control of the addicted drinker. Although Isbell describes this second kind of craving as psychological in origin, the motivational emphasis is on the internal, physical "pull" of craving, with little if any attention paid to the environmental or cognitive factors that might be involved.

What happens after craving is responded to, and the first drink is consumed? According to Jellinek (1960, pp. 145 ff.), one of the leading proponents of the disease concept of alcoholism, the first drink acts as a signal activating "the metabolism of nervous tissue cells,"

and triggers off a strong desire for additional alcohol. This desire leads to the compulsive ingestion of more alcohol, usually referred to as loss of control drinking. As Jellinek himself first put it:

> Loss of control means that as soon as a small quantity of alcohol enters the organism, a demand for more alcohol is set up which is felt as a physical demand by the drinker. . . the "loss of control" is effective after the individual has started drinking, but it does not give rise to the beginning of a new drinking bout. (Jellinek, 1952, p. 679.)

This compulsive desire to drink overrides any cognitive or volitional control; it is the complete loss of voluntary control:

> Recovered alcoholics in Alcoholics Anonymous speak of "loss of control" to denote that stage in the development of their drinking history when the ingestion of one alcoholic drink sets up a chain reaction so that they are unable to adhere to their intention to "have one or two drinks only" but continue to ingest more and more -- often with quite some difficulty and disgust -- contrary to their volition. (Jellinek, 1960, p. 41.)

Jellinek held the position that craving and loss of control were both key symptoms in "gamma" alcoholism, considered by him to be the predominant form of alcoholism

in North America. This view has been widely accepted by other proponents of the medical model (see review by Maisto & Schefft, 1977). Marconi, Fink, and Moya (1967) define loss of control as a pathognomonic symptom, "the most important clinical manifestation which usually accompanies intermittent (gamma) alcoholism and also the differential diagnosis between intermittent alcoholism and the unpathological forms of alcohol ingestion" (p. 544).

Mark Keller, the current editor of the Journal of Studies on Alcohol, has taken issue with Jellinek's notion that loss of control drinking occurs only after the alcoholic has consumed his first drink. In his 1972 paper, Keller described a second form of loss of control which he believes is the essence of alcohol addiction. Acknowledging that an alcoholic may on occasion be able to refrain from further drinking after having the first drink or two, Keller goes on to say that sooner or later he will lose control over his drinking: ". . . if an alcoholic takes a drink, he can never be sure he will be able to stop before he loses control and starts on a bout" (p. 160). Furthermore, the alcoholic, according to Keller's reformulation of the loss of control construct, has little or no control over whether he or she will take the first drink: ". . . an alcoholic does not always have the choice of whether he will take the first drink or not, or

whether he will start to drink, be it with or without the rationalization that it is to be just one drink or two. For that is precisely the nature and the essence of the addiction" (1972, p. 160).

What, then, are the factors determining the consumption of the first drink? Keller believes that certain environmental cues or stimuli precipitate a relapse:

> An addict may sometimes go about rationally debating the question, to drink or not to drink--sometimes. But not always, not consistently. His disease consists precisely in this, that at some time, under the impulsion of some cue or stimulus which may well be outside his conscious awareness, he will drink. . . . The essence of the addiction is that, when the significant cue or signal impinges upon him, though he is unconscious of it, or conscious of it but unaware of its significance, he will reach out to drink. I don't want to be mystical about these fatal stimuli or signals that enforce drinking on alcohol addicts. . . .it would go too far afield to discuss that in detail. I will therefore say here only this much: Addiction, in this conception, is thought of as a form of learned or conditioned response. (1972, p. 160).

From this perspective, it would appear that loss of control

is a kind of conditioned or "automatic" response, elicited by conditioned stimuli in the environment. Through the process of stimulus generalization, Keller believes further that almost any stimulus, including alcohol itself, can serve as a stimulus for drinking:

> For any alcoholic there may be several or a whole battery of critical cues or signals. By the rule of generalization, any critical cue can spread like the tentacles of a vine over a whole range of analogs, and this may account for the growing frequency of bouts, or for the development of a pattern of continuous inebriation. An exaggerated example is the man who goes out and gets drunk every time his mother-in-law gives him a certain wall-eyed look. After a while he has to get drunk whenever any woman gives him that look. And after a while, whenever any woman gives him any kind of look. With a bow to Woman's Lib., the sex roles may just as well be reversed. But perhaps the most important element in the spread of the addictive conditioning is that alcohol itself may become the significant cue; or it may be a particular blood alcohol level. (1972, p. 161).

Keller is careful to note that in accordance with his definition, loss of control drinking is an inconsistent symptom. Sometimes it happens, and sometimes it fails to occur:

> . . .sometimes, the loss-of-control symptom happens to be in remission. Sometimes it is in remission as a response to treatment. . . . So I have to make a special point of insisting that the loss-of-control symptom in alcoholism, like other specific symptoms in other diseases, appears only inconsistently. Sometimes it is not operating, and at such times an alcoholic may drink without tumbling into a bout. Now that I mention it--haven't we all known it all the time? (1972, p. 163).

While Keller does not evoke the concept of craving as a determinant of loss of control, his conditioning model emphasizes the involuntary, almost automatic nature of uncontrolled drinking as a response to a wide variety of conditioned stimuli. This classical conditioning approach is extended and elaborated by Ludwig and his colleagues, who reintroduce craving as a mediating determinant of relapse. Because of the complexity of Ludwig's approach, it is worth examining his position in some detail.

Craving as a Determinant of Relapse

Ludwig's early studies were based on questionnaire data. The first paper (Ludwig, 1972) reported the results of a questionnaire which was administered to 176 male patients who had completed an inpatient treatment program for alcoholism. The 161 patients who had relapsed were

asked to give their own reasons why they took their first drink after discharge from the hospital. A majority of the sample gave a variety of environmental and emotional reasons why they had "fallen off the wagon," including such factors as psychological distress (25%), family problems (13%), sociability (10%), and employment problems (5%). Ludwig notes that, "Surprisingly, only the smallest percentage of patients (1%) offer reasons akin to the subjective feeling of craving as the cause of their relapse" (p. 94). Although it is not specified in the paper, it appears that the patients were asked to provide their own reasons for relapse, in their own words, and that these responses were later classified into categories by the investigator.

In a second study, Ludwig and Stark (1974) administered a Drinking and Craving Questionnaire (DCQ) to 60 male alcoholics in an inpatient treatment setting. This time the questionnaire contained structured questions which the subjects were asked to respond to by filling out a rating scale. Among the items on the DCQ, the following question pertained directly to craving: "When you are drinking steadily, to what extent does it feel like your body 'craves' or needs alcohol?" Phrased in this form, 70% checked the rating "very much" and 15% checked "a fair amount." In another section of the DCQ it was

found that, "Although only 78% of the alcoholics reported having experienced 'craving,' 98.4% (all but one) responded when asked to 'define craving and what it means to you'" (p. 902). When the subjects were asked to define craving in their own words, 57.5% described it in terms of a need or desire for alcohol (e.g., "Have to have it"), and 42.5% defined craving as a desire for the effects of alcohol (e.g., "Don't crave the taste but the feeling"). Although there were no items on the DCQ which specifically asked if the subjects experienced craving prior to the initiation of drinking (as opposed to craving during a drinking bout or session), the authors conclude that "regardless of the particular symbolism alcoholics employ to describe the experience of craving, the common denominator of this experience is that it provides them with an acceptable excuse to resume or continue drinking" (p. 904). The data presented, however, fail to support the notion that craving may be a determinant of relapse, in contrast to craving as a desire for more alcohol once drinking has begun. In comparison with the results of the first paper (Ludwig, 1972), the second study shows that 78% of the sample will report having experienced craving -- if one asks the right questions. Curiously, there is no reference to the earlier paper in Ludwig and Stark's 1974 publication.

In 1974, Ludwig and Wikler published a paper entitled, "'Craving' and Relapse to Drink." Although no supporting data are offered, the authors describe a theoretical model which attempts to show that "craving and loss of control represent crucial determinants of relapse and excessive drinking behavior" (p. 113). As if in response to the data presented in the first study (Ludwig, 1972), the authors state conclusively that:

> Because alcoholics do not spontaneously report craving or because they offer some other reason for drinking does not necessarily mean that they are not experiencing craving or that craving is not an important determinant in the initiation and perpetuation of drinking. . . . On further probing, experimenters and clinicians will likely find an intense desire, compulsion or drive for alcohol consumption beyond the veil of the glib labels or reasons supplied by alcoholics for their drinking. (1974, p. 120.)

In a direct analogy with narcotic craving, Ludwig and Wikler consider "craving for alcohol, comparable to craving for narcotics, as representing the psychological or cognitive correlate of a 'subclinical' conditioned withdrawal syndrome" (p. 114). Based on Wikler's earlier conditioning theory of the opiate withdrawal process, the authors claim that the craving for alcohol which is assumed

to occur during an alcoholic's withdrawal from the drug can become classically conditioned to stimuli (e.g., "physical environment, drug-using or drug-dispensing associates, certain emotional states," p. 114) which are contiguous with the withdrawal experience. If an abstinent alcoholic is exposed to these same conditioned stimuli at a later date, he will experience symptoms of craving (conditioned responses) which may predispose him to relapse. In this sense, craving is similar to a "mini-withdrawal" -- a "subclinical conditioned withdrawal syndrome" as they put it. "The more frequent and severe the prior withdrawal experiences, the greater the predisposition to conditioned withdrawal symptoms with consequent desire for relief (i.e., 'craving') through drink" (p. 115).

The authors claim that craving can become conditioned to a wide variety of interoceptive or exteroceptive stimuli. In this stage of their theory, however, they go far beyond limiting these conditioned stimuli to those directly associated with withdrawal. Stimuli become capable of eliciting craving even if they are associated with drinking prior to withdrawal:

> As for exteroceptive conditioning, this would pertain to a variety of situations associated with <u>prior heavy drinking</u> or with the uncomfortable psychological and physical effects of prior withdrawal experiences. A

> conditioned withdrawal syndrome, generally subclinical in nature, with associated craving might result, therefore, whenever the alcoholic passed a bar or was in the presence of other people drinking or encountered cues relevant to previous drinking practices. (1974, p. 116, italics added.)

As with Keller's discussion of the stimulus generalization effect embracing more and more stimuli which may elicit drinking, the present analysis suggests that any cue, present during either drinking or withdrawal can become a conditioned stimulus for craving.

How does the experience of craving relate to actual drinking behavior? Ludwig and Wikler state that the relation between craving and drinking is not an invariant one. Rather, craving is viewed as a necessary but not sufficient determinant of relapse: "While the experience of craving provides an alcoholic with the necessary cognitive symbolism for goal-directed, appetitive behavior (i.e., the negative reinforcement provided by alcohol), there is no cogent reason (as with anger, hunger or sexual urge) why this subjective desire for alcohol should be directly acted upon or expressed in overt behavior, especially if there are competing drives or motivations" (p. 116). The authors describe a number of

"modifiers" of craving, including the influence of setting and other situational factors, the availability of alcohol, and the ability of the alcoholic to correctly label his physiological state of arousal as craving for alcohol. Under the proper set of circumstances, however, craving will eventuate in drinking and associated loss of control. Loss of control is described as the alcoholic's relative inability to regulate the consumption of alcohol:

> Craving is the cognitive state designating ethanol consumption as a source of relief or pleasure; it need not inevitably lead to drinking. Loss of control is the behavioral state initiated by craving and characterized by activities indicative of a relative inability to modulate ethanol consumption; it need not eventuate in gross intoxication or stupor. It may even take the form of total abstinence, when an alcoholic has incorporated the belief that he must remain abstinent because he cannot handle alcohol. (1974, p. 122).

Consumption of the first drink acts to further increase craving for alcohol, thus increasing the probability that loss of control will occur: "This first drink, then, would act like an 'appetizer,' stimulating hunger (craving, as a conditioned withdrawal response) even further because it has become sequentially conditioned to the later consumption of an 'entrée' (intoxication)" (p. 128).

An overview of Ludwig and Wikler's model of relapse is presented in Figure 2. The construct of craving appears twice in the sequence of events: first, as a conditioned response (CR) to stimuli associated with prior withdrawal experiences (CS); and second, as a response to the consumption of the first drink. Consumption of the first drink is likely to occur as a response to initial craving, given the availability of alcohol under the right set of circumstances. According to this model, loss of control drinking is triggered by the craving associated with the "appetizer" effect of the first drink.

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In an empirical test of their theory of craving as it relates to relapse, Ludwig, Wikler and Stark (1974) describe a study in which 24 detoxified male alcoholics were administered either a high or low dose of alcohol, under different labeling conditions, to determine the effects on reported craving and alcohol acquisition behavior. Half the subjects received their drinks in an appropriate labeling context (subjects consumed their preferred drink in the presence of alcohol-related cues), and half were assigned to the "nonlabel" condition (subjects consumed

ethyl alcohol with an artificially sweetened mixer in the absence of alcohol cues). Subjects were also assigned to one of three alcohol dose conditions: high dose (1.2 ml/kg of body weight), low dose (0.6 ml/kg), or a placebo group (a small amount of alcohol floating on top of the mixer). A variety of subjective, behavioral, and physiological measures was obtained at four time intervals following administration of the drinks (ranging from 20 to 200 minutes later). The authors hypothesized that the low dose of alcohol should produce a greater craving for alcohol than the high dose, because of the "appetizer" effect associated with the lower dose. Presumably, the high dose would "satisfy" the craving, and the low dose would stimulate craving for more alcohol. It was also predicted that craving and associated behaviors would be highest in the appropriate labeling condition. Subjective reports of craving were assessed by means of a "craving meter," a device that permitted the subject to indicate his degree of craving on a scale ranging from 0 to 100. A behavioral measure of alcohol acquisition was also included, in which the subject could work for alcohol on a button-pressing device. The number of button presses for alcohol was taken as the main measure of loss of control (although the alcohol could not be consumed until the end of the experimental session).

The results of the study are extremely difficult to interpret because of numerous problems in the design and analysis of the data. The groups were not independent, as all subjects received all three alcohol doses in different sessions. Because of the small number of subjects and the "highly skewed distribution and wide range of responses for craving and work measures" (p. 542), nonparametric statistics were used to analyze the data and "P values of .10 or less represented the criteria for statistical significance" (p. 542). In the absence of specific instructions to the subjects, the use of the craving meter may have biased the results because of the experimental demand characteristics involved.

The results as reported fail to support the authors' hypotheses. At the first assessment of craving, 20 minutes following the administration of the drink, the highest level of craving was reported by the placebo group in the appropriate labeling condition. Although the low dose group (appropriate labeling) showed somewhat higher levels of craving during the later assessment periods (140 and 200 minutes following consumption of drinks), it did not differ significantly from the high dose group (appropriate labeling) at any time period. In all beverage groups, the appropriate labeling condition led to significantly

higher levels of craving than the nonlabel condition -- a finding which would seem to support the role of cognitive expectancy factors rather than the physically "appetizing" effects of a low dose of alcohol. A very similar pattern of results was obtained with the operant measure of alcohol acquisition on the button-pressing task. Again, there was no significant difference in responses for alcohol for the low and high alcohol dosage groups in the appropriate labeling conditions.

In an attempt to rescue their theoretical model from the morass of conflicting data reported in the paper, Ludwig et al. introduce additional hypothetical constructs into the hopper. First, they propose that the extent of craving is limited by a "hypothetical blood alcohol ceiling" -- a kind of upper limit of intoxication that differs from individual to individual. Second, the setting in which the first drink is consumed and the mental set of the alcoholic may impose additional "cognitive ceilings" against the expression of craving:

> . . .we propose that within a physical setting or exteroceptive situation conducive to natural cognitive labeling the degree of craving should be a direct function of the difference between the actual blood alcohol level attained by the first drink and the hypothetical blood alcohol ceiling for each alcoholic. In the

> presence of inappropriate or incongruous settings, not conducive to natural cognitive labeling, the magnitude of craving should be substantially less than under all appropriate exteroceptive conditions, regardless of actual alcohol dose. This is largely because the hypothetical ceiling for desired blood alcohol levels will more likely be determined by individualized mental constraints (i.e., fluctuating "cognitive ceilings") against the expression of craving and alcohol acquisition in incongruous settings. (pp. 545-546.)

According to Ludwig and his co-workers, the concept of craving is paramount to the understanding of the relapse process. It would appear to be a very global and all-encompassing phenomenon indeed, since it can be elicited by a wide variety of conditioned stimuli, ranging from cues associated with drinking to those associated with withdrawal from alcohol. The conditioning can also occur with both interoceptive and exteroceptive stimuli, including almost any state of physiological arousal: ". . .since anxiety, nervousness, jitteriness, and other types of emotional dysphoria may produce such physiological responses as increased heart rate and respiration, tremulousness, autonomic lability, increased sweating, insomnia, all of which represent changes associated with the alcohol withdrawal syndrome, we should anticipate that these states,

induced by either arguments with spouses, employment difficulties or loneliness, may likewise evoke craving" (Ludwig, Wikler & Stark, 1974, pp. 539-540). Craving is certainly not limited by the authors to situations associated with acute withdrawal from alcohol -- otherwise we would expect the greatest craving to occur in such settings as alcoholism detoxification and treatment centers. Unfortunately, the experience of craving does not always lead to relapse or loss of control drinking, because of the influence of numerous "modifiers," such as the setting, appropriate labeling conditions, the "subclinical" intensity of the craving symptoms, and the restraints imposed by both the hypothetical blood alcohol ceiling and the fluctuating cognitive ceiling. And even if craving is elicited, despite these various constraints, it may not be recognized or labeled correctly by the alcoholic because of its similarity to a variety of other arousal states. The explanatory power of the craving construct seems to far outweigh its ability to predict behavior.

Craving: A Critique

Let us return to the case study presented earlier, to see how the theoretical model of craving might be used as an explanation of Liane's relapse. It is difficult to see how craving could have served as a determinant of her first drink on July 17. To the best of my knowledge, Liane

had never experienced withdrawal symptoms in a restaurant setting, so there were no stimuli present in that situation to elicit a conditioned craving response. Her luncheon companion was not someone she drank with on a regular basis, and it is unlikely that Sharon alone could have served as a cue for craving. Did the consumption of the first drink serve as an "appetizer" for more alcohol? This seems most unlikely, as it took Liane almost an hour to finish her drink. No loss of control drinking occurred that day as a result of taking the first drink, and she was able to maintain total abstinence for an additional twenty days until she repeated the luncheon drinking experience on August 6. Once again, there was no report of craving, and no loss of control drinking occurred.

It was not until August 9, 23 days following her first drink, that she drank to the point of intoxication. Prior to drinking that day, Liane reported that she felt anxious and upset about being alone. It is possible, of course, that she experienced craving that afternoon (elicited by the cues present in her apartment, where she used to engage in heavy drinking), and that she mislabeled this feeling as loneliness and anxiety. But why would the cues associated with prior heavy drinking and/or withdrawal elicit a conditioned craving response on that <u>particular</u>

day, the 81st day she had spent in the apartment since her discharge from the hospital? It seems more likely that Liane did label her feelings correctly, and then sought a remedy for her anxiety and tension: she tried to find some tranquilizers. Unfortunately, in her unsuccessful search for the tranquilizers, she accidentally discovered some alcohol. After considerable hesitation, she drank the first scotch, probably hoping that it would exert a tranquilizing effect. When she "didn't feel anything at first " (no reduction of anxiety, no craving), she took the second drink. It was as though she sought a certain effect from the alcohol (reduction of anxiety), and when she failed to obtain it after the first drink, she decided to increase the dosage. Rather than an involuntary response to the physical demand of craving, her drinking seemed to be determined more by the cognitive expectation of the effects of alcohol. We will return to elaborate this important difference in a later section of the paper.

It should be clear by now that from a scientific perspective, craving is a superfluous construct, and the hypothesis that craving is a crucial determinant of relapse is a basically untestable tautology. As Mello (1975) has also noted in her review of craving and loss of control, to say that craving is a determinant of drinking is to engage

in circular reasoning: craving is defined by the very behaviors it is evoked to explain. Because craving is described as a subjective, internal "subclinical" state, its presence can only be verified objectively in terms of the behavior it is supposed to determine -- namely, loss of control drinking. Craving is a superfluous construct because it presumably can be elicited by almost any stimulus associated with prior drinking or withdrawal, subject to the restraint of a wide variety of modifiers. As such, it has almost no utility to predict behavior. Craving has become reified as an apparent explanation of relapse within the medical model of alcoholism; it has become a convenient catch-all construct to attribute as the "cause" of relapse, much as "will-power" has been reified as the apparent "cause" of continued abstinence.

To say that an alcoholic drinks <u>because</u> he craves alcohol is as circular as to say that an individual eats <u>because</u> he is hungry, or that a person trembles <u>because</u> he is anxious. In each case, the existence of the internal state is inferred from observation of the very behavior it is supposed to determine (see Sarbin, 1968, for a parallel analysis of anxiety as a "determinant" of behavior). Craving is not a "cause" of relapse or loss of control. Unless it can be defined operationally by specifying

observable criteria (i.e., physiological or biochemical measures) which are independent from the behavior it is supposed to explain (relapse or loss of control), craving is best thought of as a hypothetical construct. From a scientific standpoint, the construct of craving serves only to distract the investigator in the search for independent variables which actually determine drinking behavior.

There are a number of undesirable consequences of teaching an alcoholic to believe that relapse or loss of control is somehow "caused" by craving. If the abstinent alcoholic "slips up" and takes a drink, he or she can then attribute the cause of the drinking to craving, an internal physical state over which the alcoholic has no control. Whether craving is construed as a "symptom" of the underlying addiction or disease, or as an elicited "conditioned response," the emphasis is on the involuntary nature of the reaction. Loss of control drinking becomes an inevitable consequence of this self-fulfilling prophecy. The alcoholic is absolved of any personal responsibility in the relapse process -- he becomes a helpless victim of the symptomatology of his disease, over which he has no control. It is easy to see how this belief could serve as a convenient excuse for drinking.

Should we do away altogether with the concept of craving? Despite the arguments advanced by Ludwig and his colleagues,

the concept of craving seems to have little or no scientific utility as a predictor of relapse or loss of control drinking. Use of the term should be limited to phenomenological descriptions of the subjective desire for alcohol experienced by addicts undergoing withdrawal. In this context, it would be more accurate to speak of craving as a strong desire for the alleviation of unpleasant withdrawal symptoms: craving for the relieving effects of alcohol, rather than craving as some kind of internally based cellular "need" for alcohol itself. Although there is no scientific evidence to support the assumption that craving is an important determinant of relapse, the fact that some alcoholics believe this to be the case may have important effects on their behavior. The role of personal beliefs and expectancies as they affect relapse will be discussed shortly.

In recent years, a number of experimental studies have been published which have challenged the validity of both craving and loss of control as determinants of relapse or uncontrolled drinking (i.e., "binge" or "spree" drinking). Many of these studies have involved the direct observation of the drinking behavior of alcoholics in controlled settings. Since these studies have recently been reviewed in detail by other authors, the interested reader may wish to consult these sources (e.g., Maisto,

Lauerman & Adesso, 1977; Maisto & Schefft, 1977; Mello, 1975; Pomerleau, Pertschuk & Stinnett, 1976) for further information. As is clear from an examination of the recent experimental literature, the constructs of craving and loss of control as key concepts in the disease model of alcoholism have received little or no empirical support.

The Relapse Process: A Cognitive-Behavioral Analysis.

Situational Determinants of Relapse

According to the basic assumptions of the medical model of alcoholism, relapse is an all-or-none event. Abstinence is the indication of treatment success, and relapse is an indication of treatment failure. From this perspective, the particular details associated with the relapse (the specific antecedents, time, situation, personal feelings and emotions involved, etc.) are of little importance; the emphasis is on the "emergence" of the key symptom of the "underlying" addiction or disease, loss of control drinking. To relapse implies that one has "lapsed" back into the disease, succumbing to the influence of internal, biological factors beyond the individual's control. As a symptom of an underlying disease, loss of control drinking is no different from other symptoms: it is treated much like a fever, a symptom which emerges from within, acting as a sign that the disease has "broken out" again.

A review of the literature dealing with the environmental or situational aspects of relapse reveals only a few studies to date. In a paper describing the characteristics of the relapse situation of alcoholics treated with aversion therapy, Burt (1974) reported that about 80% of his sample (30 men and 4 women) took their first drink in a location which differed from their preferred location prior to treatment, but no detailed descriptions of these locations are given in the paper. Hore (1971) followed a group of 22 alcoholic patients receiving treatment in an outpatient clinic for periods ranging up to six months. He attempted to relate relapses in this group to significant life events reported to occur by the patients in the same general time period. Hore analyzed the events associated with relapse (41 episodes of relapse and 52 significant events), and assigned them to the following categories: (a) Personal interaction, or disturbances in an emotional relationship (33%); (b) Work events, involving a change in the patient's working life (33%); (c) Events involving a health change in the patient or in members of the family (20%); and (d) Events involving a change of residence (13%). Gregson and Taylor (1977), among others, have attempted to isolate factors which are predictive of relapse, such as degree of cognitive dysfunction, membership in AA,

and selected demographic data, but no data are reported on the situational or intrapersonal factors immediately preceding the relapse.

I first became interested in looking at the situational factors associated with relapse while I was engaged in a study to determine the effectiveness of electrical aversion as a treatment procedure for alcoholism (Marlatt, 1973). To increase the generalization of the aversion effects, we conducted the treatment sessions in a simulated bar, assuming that the contextual cues of the drinking setting would also acquire aversive properties through the principal of high-order conditioning. To assess the effectiveness of this procedure, I decided to gather as much information as I could about the particulars of the relapse situation for those patients who took at least one alcoholic drink during the follow-up phase. By doing so, I hoped to determine whether the aversion procedures had any generalization effects in terms of the setting in which relapse occurred.

The patients in our sample consisted of 65 males, most of them chronic alcoholics drawn from a rural catchment area. During the first ninety days of the follow-up period, 48 subjects in the sample consumed at least one drink. Each of these patients was personally interviewed within a few weeks of the relapse episode as part of our

follow-up evaluation. We asked a variety of questions about the events leading up to the consumption of the first drink, soliciting information about the location, time of day, presence of others, characteristics of the drinking situation, external or environmental events occurring in that general time period, and the patient's reported feelings and emotions on the day of relapse. We then attempted to sort the list of relapse situations into independent, operationally defined categories. Raters were trained to assign specific episodes to categories until they achieved at least 80% interrater agreement. The results of this analysis are reported in Table 1.

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Insert Table 1 About Here

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It is clear from the results presented in Table 1 that all of the relapse cases could be assigned to relatively few categories. The first two categories, accounting for over half the cases, usually involved an interpersonal encounter. Most of the situations in the first category (29%) involved an episode in which the patient was frustrated in some goal-directed activity (typically by another person who criticized or negatively evaluated him), and reported feelings of anger. Rather than expressing this anger overtly, however, the patient ended up taking a drink.

In the second category (23%), the patients reported being unable to resist either direct or indirect attempts by others to engage him in drinking (social pressure). The other two major categories, accounting for about a third of the cases, were basically intrapersonal in nature. Temptation situations were quite common (21%), although it is possible that the sudden "urge" to drink was determined by other environmental or emotional factors which were not identified in the assessment interview. Negative emotional states (10%), including feelings of depression, anxiety, and boredom, accounted for only relatively few relapse episodes.

How limited are these findings? If the results applied only to our first sample, then the analysis would provide little more than a list of idiosyncratic relapse situations, possibly masking a more central underlying determinant of relapse, such as craving for alcohol. A recent follow-up study with another sample of male alcoholics ($\underline{n}$ = 25), revealed that the same categories accounted for almost all of the relapse episodes, although the percentage of cases in each category differed from the first sample: frustration and anger, 16 %; social pressure, 17 %; intrapersonal temptation, 15 %, negative emotional state, 43 %; and miscellaneous other, 9 % (Chaney, O'Leary & Marlatt, 1977).

In a related series of studies, we have also been

investigating the extent to which the key features of the relapse situations act as determinants of alcohol consumption in nonalcoholic drinkers. Using heavy social drinkers as our subjects, we have manipulated situational and emotional variables drawn from our analysis of relapse episodes to determine how such factors affect drinking behavior in standardized alcohol consumption tasks (cf., Marlatt, 1977). These studies have shown that feelings of anger (Marlatt, Kosturn & Lang, 1975), anticipation of interpersonal evaluation (Higgins & Marlatt, 1975), and modeling as a form of "social pressure" to drink (Caudill & Marlatt, 1975) all serve as powerful elicitors of increased drinking behavior in nonalcoholic subjects.

Based on this accumulating evidence, it seems clear that environmental/situational factors, and the emotional and cognitive reactions which accompany them, are important determinants of relapse. While other factors may also play a role in the relapse process, such as demographic and other individual differences, past drinking history, degree of cognitive impairment, and the type of treatment received, it is the effect of these high-risk situations and the individual's response to them that is most closely associated with the occurrence of the relapse itself. If the alcoholic does not know how to cope with these high-risk situations when they occur in the natural environment, the

probability of relapse will increase.

Expectancies About Alcohol and Its Effects

The evidence reviewed in the last section suggests that the probability of relapse increases in high-risk situations, particularly for those individuals who lack the appropriate skills to cope with the problem. In the latter instance, alcohol represents the only "coping" alternative available. For most alcoholics, drinking has become the predominant, overlearned, and habitual response to stressful situations. Over years of heavy drinking, alcohol begins to acquire the properties of a potent elixir, to turn "lows" into "highs," an image which is constantly reinforced by the romantic display of liquor and drinking presented in advertising and the media.

What is there about alcohol that makes it such a powerful reinforcement? The experimental literature on the reinforcing effects of alcohol in human subjects has yet to provide an agreed-upon answer to this question. The original hypothesis, that alcohol is reinforcing because it reduces tension and anxiety, has failed to receive consistent support in recent studies (see reviews by Cappell, 1975; Marlatt, 1976). This issue is complicated by the fact that the physiological response to alcohol appears to be biphasic in nature, and that the affective

consequences of drinking depend on the dosage consumed:

> There is increasing evidence to suggest that alcohol, at least in small doses, has a stimulating effect on physiological arousal. Emotional responsiveness to alcohol may be biphasic in nature, with the depressant or sedative effects occurring only at higher doses. The increase in arousal occurring at relatively low blood-alcohol levels may be one of the motivating factors in social drinking: people often say that they drink to get high or to experience a lift, not to experience a low emotional state. A person who returns home after a hard day's work and consumes a couple of cocktails before dinner may experience a physiological boost that temporarily relieves his state of fatigue or physical exhaustion. The drinker, however, may label this effect as one of relaxation instead of excitation. (Marlatt, 1976, p. 290.)

Whatever the physiological effects may be on the brain and nervous system, it is the drinker's subjective experience of these effects that determines his personal attitudes and beliefs about alcohol. Like many drugs which have variable and nonspecific effects on emotional and physical response systems, the consumption of alcohol produces a range of ambiguous effects which can be interpreted differentially by the drinker, depending on various

personal and situational factors (cf., Schachter, 1964). To take but one example, Pliner and Cappell (1974) have shown that the presence or absence of social companions can influence subjects' evaluation of the effects of alcohol. Social drinkers who consumed alcohol in a group setting interpreted its effects in terms of changes in mood and emotional state, whereas solitary drinkers described the effects solely as changes in physical symptoms. The influence of set and setting will often determine whether a particular drinking experience is evaluated as pleasant or unpleasant by the individual drinker.

More research needs to be done to pin down the effects of alcohol on cognitive processes in human subjects. At the present time, we know almost nothing about the effects of varying doses of alcohol, consumed in qualitatively different settings by subjects who differ in terms of their drinking histories, upon subjective states of consciousness. In one of the few investigations in this area, McClelland and his associates conducted a series of studies dealing with the effects of alcohol on cognitive fantasies and ideation in male drinkers (McClelland, Davis, Kalin & Wanner, 1972). Personal fantasies were assessed after consumption of alcohol with use of the TAT, a projective story-telling test. Although the research suffers from a

number of methodological flaws, the results are provocative enough to warrant further investigation. The major conclusion drawn from the studies was that alcohol facilitates or stimulates a sense of "power" in male social drinkers. With relatively low doses of alcohol, the fantasies centered around themes of "social power," or power for the good of others or for a cause. At higher dose levels, the fantasies were more associated with themes of "personal power" -- less altruistic, and more self-aggrandizing in nature. If alcohol acts to increase the drinker's sense of perceived power or control, at least at the cognitive level, it seems likely that the attractiveness of alcohol as a coping strategy would increase in situations where the individual feels powerless or otherwise lacking in personal control (Marlatt, 1976).

In addition to the reinforcing subjective effects of alcohol, a case can be made for the hypothesis that alcohol consumption provides a convenient excuse for the execution of otherwise unacceptable forms of behavior (cf., Sobell & Sobell, 1973). An individual can avoid personal responsibility for engaging in certain antisocial acts, for example, if he attributes the cause of the behavior to the disinhibiting effects of alcohol. "I was drunk, and I didn't know what I was doing," goes the typical excuse. Recent

research has supported the idea that cognitive processes and expectation effects outweigh the physiological effects of alcohol in facilitating both aggressive behavior (Lang, Goeckner, Adesso & Marlatt, 1975), and sexual arousal (Wilson & Lawson, 1976) in male social drinkers.

The importance of cognitive expectancies about alcohol and its effects was highlighted for me by the results of a study we conducted on the determinants of loss of control drinking (Marlatt, Demming & Reid, 1973). In this experiment, both nonabstinent male alcoholics and matched social drinkers were asked to take part in a taste-rating task, in which they were asked to sample and compare the taste qualities of either alcoholic or nonalcoholic beverages. The taste-rating task served as an unobtrusive measure of consumption, as subjects were free to sample as much of each drink as they wished to in making their ratings (see Marlatt, 1977, for a full description of the taste-rating task procedure). In order to control for both the physiological effects of alcohol and the subject's expectancy or belief about the effects of alcohol, we employed a diacritical factorial design with subjects assigned to one of four independent conditions. Half the subjects were told they would be rating drinks containing alcohol (vodka and tonic), and half were told they would be rating nonalcoholic beverages (tonic water only). At the same time, we independently

varied the alcoholic content of the drinks in such a way that half the subjects in each of these two conditions actually received drinks containing vodka, and half received drinks containing only tonic water. We had found on the basis of pilot studies that a drink containing one part of vodka to five parts of tonic could not be reliably identified as containing alcohol on a better than chance basis. Prior to the taste-rating task, all subjects were given a "primer" dose of the drink they would be sampling, to determine if the alcoholic subjects receiving alcohol would show loss of control or increased consumption in the drinking task.

The results of the study were clear cut. The only significant determinant of overall beverage consumption, and subjects' later estimates of the alcohol content of their respective drinks, was the expectancy factor. Regardless of the actual alcohol content of the drinks, both alcoholic and social drinker subjects consumed significantly more beverage if they believed they were sampling drinks containing vodka. Similarly, subjects assigned to the told tonic/receive alcohol condition consumed relatively little beverage, whether or not the drinks actually contained vodka. This experimental design is superior to the traditional two-group design (alcohol administration vs.

placebo), because it controls for the expectancies of receiving both alcoholic and nonalcoholic beverages. The results of the study provided evidence that cognitive mediational factors must be taken into account in any investigation of the effects of alcohol on human behavior. Other investigations support the finding that prior expectations exert a stronger influence than the physiological effects of alcohol for a variety of behaviors in both alcoholics and social drinkers (Engle & Williams, 1972; Lang, et al., 1975; Maisto, et al., 1977; Wilson & Lawson, 1976).

If expectations are based on past experiences with the effects of alcohol, why doesn't the alcoholic maintain a negative expectancy about drinking? Most of the long-range effects are predominantly negative, including both the physical symptoms of excessive consumption (nausea, hangover, and withdrawal) and the personal and social consequences of problem drinking (domestic strife, employment difficulties, and the like). Despite repeated exposure to these unpleasant consequences, the fact remains that these negative effects are delayed in time, relative to the immediate positively reinforcing effects -- the initial "high" of the biphasic response to alcohol. In addition, the alcoholic's memory of the long-range effects may be

impaired because of blackouts and state-dependent learning effects (cf., Overton, 1971). In a series of studies by Mendelson and his colleagues (e.g., McGuire, Mendelson & Stein, 1966; Tamerin, Weiner & Mendelson, 1970), alcoholics were asked to describe their initial expectancies about the effects of alcohol on their feelings and moods prior to a scheduled period of drinking in a research ward setting. Almost all the subjects anticipated that alcohol would make them feel more relaxed, more comfortable, and less depressed. These expected effects were directly opposed to the actual dysphoric effects of prolonged drinking reported by the subjects during the intoxication phase of the study.

The expectation of positive effects, combined with the initial reinforcing influence of alcohol itself (the immediate stimulation of the "high"), act together as potent sources of motivation to take the first drink. The desire for the anticipated reinforcing effects of drinking is so strong that it is understandable that some alcoholics speak of "craving" for alcohol -- much as someone else might speak of an overwhelming craving for hot fudge sundaes or pecan pie. In this sense, to crave is to desire the anticipated pleasures of consumption; <u>wanting</u> to experience the pleasurable effects of a food or drug is not the same thing

as experiencing an internal need for the substance itself.

The Abstinence Violation Effect

The combination of positive expectancies for the effects of alcohol and the stress of a high-risk situation greatly heightens the probability of relapse. What happens when the abstinent alcoholic responds to these forces by taking a drink? How does the consumption of the first drink influence the probability of taking the second drink or drinking to the point of intoxication? Why is it more difficult to return to a position of complete abstinence following the experience of a single slip? What is there about a "relapse" that makes an alcoholic "lose control" over subsequent drinking?

Our previous review of the assumptions of the disease model provided one answer to these questions: that the consumption of one or two drinks elicits an addictive craving for more alcohol, triggering an uncontrolled bout of excessive drinking. But the phenomena of "relapse" and "loss of control" extend far beyond the alcoholic and his reputed disease. In my clinical practice (and in my various attempts to control my own behavior), I have experienced the same chain of events associated with other behaviors, particularly smoking and eating. The following examples will illustrate the point. (a) A young man decides to quit

smoking. After a month of abstinence, he smokes one cigarette at a party. Within two weeks, he is smoking at the same rate he smoked prior to quitting. (b) A young woman goes on a strict diet to lose weight. During one afternoon when she is feeling depressed, she goes out and treats herself to a banana split. Afterwards, she feels that because she "blew" her diet, she can eat anything she wants to until the next diet begins. In both examples, the behavioral sequence is the same: the individual commits himself or herself to an indefinite period of abstinence or strict control over patterns of consumption. Then, for whatever reason, the taboo behavior occurs. The probability of repeating the behavior increases dramatically, and a full-blown "relapse" ensues. Are we then entitled to conclude that the ex-smoker, the dieter, and the abstinent alcoholic are all victims of an underlying, latent addictive disease which can be triggered by one cigarette, one ice-cream sundae, or one drink into compulsive craving and loss of control consumption?

To account for the similarity of the relapse process across different consummatory behaviors, we have postulated a common cognitive denominator: the Abstinence Violation Effect (AVE).[3] The AVE is postulated to occur under the following conditions: (a) The individual is

personally committed to an extended or indefinite period of abstinence from engaging in a specific behavior; (b) The behavior occurs during this period of voluntary abstinence. The intensity of the AVE will vary as a function of several factors, including the degree of commitment or effort expended to maintain abstinence, the length of the abstinence period (the longer the period, the greater the effect), and the importance or value of the behavior to the individual concerned. We hypothesize that the AVE itself is characterized by two key cognitive elements:

(1) A cognitive dissonance effect (Festinger, 1957, 1964), wherein the occurrence of the previously restricted behavior is dissonant with the cognitive definition of oneself as abstinent. Cognitive dissonance is experienced as a conflict state, and underlies what most people would define as guilt for having "given in to temptation."

(2) A personal attribution effect (cf., Jones, Kanouse, Kelley, Nisbett, Valins & Weiner, 1972), wherein the individual attributes the occurrence of the taboo behavior to internal weakness or personal failure (e.g., to "lack of will power" or "insufficient personal control" over one's behavior), rather than to external situational or environmental factors.

Although the dissonance and attribution components of

the AVE may overlap to a degree, the additive effects of both reactions will greatly increase the probability of repeating the restricted behavior, according to our theoretical model. The dissonance state will vary in intensity depending on the duration of the abstinence period and the degree of personal and public commitment to the role of an abstainer. A member of Alcoholics Anonymous, for example, who refers to him/herself as a "recovering alcoholic," and has made a public pledge never to take another drink, is particularly susceptible to an intense dissonance reaction upon consuming a single drink. According to classical cognitive dissonance theory, dissonance is experienced as a negative emotional drive state, and can serve as a motivating force to engage in behaviors (or changes in cognition) which serve to reduce dissonance. Consumption of more alcohol, following the first drink, is a particularly effective behavior in this regard, because (a) to the extent that alcohol reduces tension or anxiety, it should also be effective in reducing feelings of dissonance or guilt; and (b) the alcoholic can modify his self-image to achieve consonance with his drinking behavior (e.g., "I guess I still haven't fully recovered; the disease has got me again"). From the perspective of dissonance theory, it is very difficult to "undo" the act of taking the first

drink: a single drink is enough to shatter the image of oneself as an abstainer.

Most abstinent alcoholics are justifiably proud of their continued sobriety. Although they may partially attribute their success to a specific experience, such as involvement in a particular treatment program or in A.A., it seems likely that they also feel personally responsible for having taken the "first step" towards a new life, totally free from alcohol. As each day of abstinence passes, "one day at a time," their self-confidence and feelings of personal control over the temptations to drink increase. How is this self-image affected by taking the first drink? To the extent that the individual feels personally responsible for "giving in" and consuming that drink, attribution theory would predict that he or she would attribute this "failure" to internal, personal causes (cf., Weiner, Frieze, Kukla, Reed, Rest & Rosenbaum, 1971). Even though an objective observer of the relapse might ascribe the causes to environmental stressors beyond the individual's control, the individual concerned will frequently attribute the event to personal weakness or other shortcomings. People often draw inferences about their own internal states through the observation of their behavior (Bem, 1972). Thus, if a relapse occurs, the alcoholic is likely to infer

a lack of will power or personal control as the determinant of the relapse. If the relapse is viewed as a personal failure in this sense, the individual's expectancy for continued failure will increase as a result (Weiner, et al., 1971). An expectancy of failure is likely to mediate decrements in actual performance. If one feels weak-willed and powerless for giving in to the temptation of the first drink, the expectation of resisting the second or third drinks is correspondingly lower: "once a drunk, always a drunk."

From this perspective, loss of control drinking is determined in large part by the alcoholic's perception of having "lost control" when the relapse first occurred. By attributing the relapse to personal failure, the absence of will power or strength, the alcoholic relinquishes control to alcohol. Attributing the relapse to an underlying disease or addiction has much the same effect: the alcoholic still believes he is personally helpless in the face of internal forces beyond his control. The relapse is still "caused" by internal, intrapersonal factors -- whether they are labeled as symptoms of a disease state, or as signs of personal weakness.

The conditions for a full-blown relapse are as follows: (a) The abstinent alcoholic feels "in control"

until he encounters a high-risk situation which challenges his perception of control; (b) The individual lacks an appropriate method of coping with the high-risk situation, or fails to engage in a coping response; (c) He has positive expectancies about the effects of alcohol, and alcohol is available; (d) He takes the first drink; (e) He experiences one or both components of the Abstinence Violation Effect (cognitive dissonance and/or attribution of failure to internal weakness); (f) The probability of continued drinking markedly increases. Whether or not the individual will continue to drink immediately following the consumption of the first drink will depend on a number of factors, including the intensity of the AVE and the effect of situational constraints (presence of observers, time limitations, availability of alcohol, and other factors). Consumption of the first drink shifts the probability of subsequent drinking upwards, to a much higher level than it was during the abstinence period.

Loss of Control Drinking Revisited

If "loss of control" drinking does occur following consumption of the first drink, how is this behavior explained? We have already noted that consumption of the first drink increases the probability of subsequent drinking. The specific form of this behavior may vary,

depending on a number of factors. It may be possible, for example, that alcoholics can be explicitly trained to exert control upon their subsequent drinking, as has been demonstrated recently by some behavioral investigators (esp. Sobell & Sobell, 1973 , 1976). Treatment interventions to modify the typical response to relapse will be discussed in the final section of this paper.

The precise form of behavior traditionally associated with loss of control drinking has never been clearly specified in the literature. From descriptions which are available (e.g., Jellinek, 1960), usually drawn from retrospective reports given by alcoholics or from the A.A. literature and lore, one gets the picture of a crazed alcoholic compulsively driven by addictive craving, gulping down drink after drink of whiskey until he falls on the floor in a drunken stupor. Unfortunately, this behavior has never been reliably demonstrated in any observational studies of alcoholics' drinking behavior (Mello, 1975). Although these reports certainly do show that alcoholics maintain a very high blood-alcohol level during periods of drinking, they fail to provide support for the notion of loss of control drinking as a compulsive, uncontrolled behavior.

One can only infer that control has been "lost" by observing the drinking behavior itself -- a tautology similar

to the circular definition of craving reviewed earlier. A strict operational definition would omit any reference to the construct of control, and would focus on the rate of drinking itself (sip rate and amount consumed) and the resultant blood-alcohol level. There are a number of factors which may account for the high rate of drinking associated with the loss of control phenomenon:

(1) Absorption of alcohol rate. It takes a certain amount of time for alcohol to be absorbed into the blood stream and distributed throughout the brain and nervous system before its effects can be perceived by the drinker. This time period varies as a function of the subject's sex and weight, amount of undigested food in the stomach, dilution of the alcohol in the drink consumed, and other such factors, but it is frequently estimated to take from 15 to 30 minutes before the effects are "felt" (cf., Kalant, 1971). Because of the alcoholic's strong positive expectancies about the effects of alcohol, the rapid rate of beverage intake may reflect his "impatience" to experience the desired effects during the absorption delay period. From the standpoint of reinforcement theory, the high response rate indicated by gulping down a number of drinks in quick succession resembles the burst of responses typically observed in subjects exposed to a Variable Interval (VI)

schedule of reinforcement. In a VI schedule, reinforcement occurs at variable time intervals, and is associated with a very high response rate prior to reinforcement (e.g., Verhave, 1966). The alcoholic may show a similar burst of responses during the absorption period until the alcohol begins to "hit" him.

(2) Tolerance effects. Alcoholics typically show an acquired tolerance for alcohol, such that a progressively increased dosage of alcohol is required to obtain the same degree of effect in a given individual over time (Kalant, Leblanc & Gibbins, 1971). While this tolerance effect may decrease over a prolonged period of abstinence, it still must be considered as a factor in determining the high rate of consumption associated with loss of control drinking.

(3) Overdose effects. Because of the combined influence of the absorption delay and tolerance effects, the alcoholic may frequently exceed a desired blood-alcohol level (associated with the optimal "high") by overdosing himself during the period of rapid intake. Although the literature contains little or no mention of the blood-alcohol levels considered optimum by various alcoholics, let us consider a hypothetical example. Suppose an alcoholic desires to experience the "high" effects associated with a blood-alcohol level in the .05% to .07% range. Under

normal circumstances, this level would be achieved approximately 30 minutes or so after the consumption of about two or three drinks. If he tosses down the first drink within a minute or so, and nothing happens, he is likely to consume another drink in short order. Within the space of 20 minutes, a number of additional drinks could be consumed before the effects of the first drink or two are experienced. As a result of this overdose, the ascending limb of the blood-alcohol curve rises quickly past the desired level and may peak in the intoxication range (greater than .10%). Direct observation of the drinking behavior of alcoholics reveals that they frequently do drink in this rapid manner (Sobell, Schaefer & Mills, 1972). The high blood-alcohol level resulting from the overdose is likely to elicit the range of dysphoric effects associated with high dose levels. The reinforcing "high" experienced at lower blood-alcohol levels is superseded by the unpleasant "low" of an overdose (cf. the biphasic response to alcohol with increasing dosage). The alcoholic may harbor the expectation that consuming even more alcohol would alleviate these unpleasant effects, and a vicious circle of drinking may ensue.

Overview of the Relapse Process: Return to the Case Study

A schematic representation of the cognitive-behavioral

analysis of relapse is presented in Figure 3. In reviewing the main points of this analysis, let us return to the case study of Liane's relapse presented at the beginning of this paper. Liane was abstinent for 57 days following her release from the hospital. During this period, she saw herself as "in control" and became increasingly confident of her ability to maintain abstinence as the days passed. She soon felt confident enough to establish a new social relationship, and invited her neighbor Sharon out to lunch. It is significant that Liane played the role of hostess during this encounter: she took the initiative to ask Sharon out, and she paid for the lunch. Prior to arriving at the restaurant, Liane was in control of the situation ("Both of us were dressed up, and I felt very at ease, cheerful and confident"). Suddenly, a high-risk situation developed, and she ended up taking her first drink.

There are a number of factors which contributed to this high-risk situation: (a) This was Liane's first venture into the social realm, and she was with someone she did not know well. Although she wanted to become friends with Sharon, Liane did not yet feel comfortable in telling her about her past drinking problems. Had Liane disclosed this information previously, she would have had little difficulty in dealing with the cocktail waitress when she came to take the order;

(b) The entire social occasion was a challenge to Liane's confidence, because of her general lack of social skills. Through the influence of liquor advertising and the romanticism associated with drinking as presented on television and the media, Liane had developed an image of herself as secure and poised in social settings -- provided she had a drink in her hand ("I was especially vulnerable to the unreality of alcoholic romanticism used in advertising, television, and movies. . . .this romanticism is a stimulus for envy and anxiety in me"); (c) Liane had not prepared herself for the encounter with the cocktail waitress. Had she anticipated this event, she could have avoided it altogether (by choosing a restaurant without a liquor license), or she could have come up with some excuse and ordered either nothing or a nonalcoholic beverage. As it turned out, Sharon ordered her drink first, thus providing a powerful modeling cue for Liane. In Liane's eyes, Sharon was a prestigious model: she was older, married, and she could drink with impunity. What else could Liane do? After all, she was the hostess, and supposedly in control of the situation. "'Gin and tonic, please,' just came out. I didn't plan it and I wasn't prepared for it." The first drink was taken.

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Insert Figure 3 About Here

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As indicated in Figure 3, if Liane had managed to cope with the high-risk situation by engaging in any of the constructive alternative behaviors described above, she would have reinforced her sense of perceived control and confidence in handling similar situations. The abstinence period would then continue, and her perception of her ability to cope successfully with future problematic situations would grow in strength. Unfortunately, she was unable to cope with the situation, and ordered the drink instead. Her reaction to the first drink appears consistent with the Abstinence Violation Effect described earlier. From the first sip, her response is characterized by conflict and guilt: "The first sip was so strong, it literally shocked me. It was difficult for me to finish the drink. I am not entirely sure whether or not I imagined some of the physical effects, perhaps because of guilt, or if it was real." It seems most unlikely that a single mixed drink, consumed slowly during the course of a 45-minute luncheon, would produce much of a physical effect under normal circumstances. The intensity of Liane's AVE is experienced as a combination of physical and psychological effects ("I got a headache, my nose got stuffy, my thoughts were tangled and my voice and actions were abrasive"). Although she did not order a second drink, probably due to the constraints of the particular

social situation and the intensity of the unpleasant reaction, she had nonetheless crossed over the line between abstinence and drinking.

Our model predicts that the probability of subsequent drinking will increase following the experience of the first drink. About three weeks later, Liane again took Sharon out to lunch, and again ordered a drink. On this second occasion, she admitted that she had considered having the drink in advance ("at least I had no real intentions of not having that drink"). Perhaps she felt an increased sense of confidence because she had not "lost control" after the experience of having the first drink. Nevertheless, she again felt the pangs of the AVE: "I didn't feel good about having that drink. It lowered my self-esteem and did create some guilty and remorseful feelings." The stage was now set for the full relapse to occur.

It happened just three days later. Again, Liane was faced with a high-risk situation. She faced a lonely Saturday evening at home, and she had "no plans for doing anything that night." Her loneliness increased during the afternoon, facilitated by the moods evoked by listening to her records. Late in the afternoon, her romantic fantasies increased and she began to feel increasingly sad and anxious. At this point, Liane felt that she might be able to cope effectively

with her feelings by taking a tranquilizer. In her unsuccessful search for tranquilizers, she discovered the miniature bottles of scotch and the beer belonging to her parents.

Liane had failed to cope successfully with the situation. Knowing that she would be alone, she could have made alternative plans to go out for the evening, spending time with her parents or friends, or going to a movie. Another point of intervention arose when she began to feel anxious about being home alone. Had she been trained in a method of relaxation, such as muscle relaxation or meditation, she might have calmed herself down and considered other activities to occupy her time. Instead, Liane reverted to her old pattern of coping, and sought out the tranquilizers. Not being able to find any, she became even more upset. At that point, finding the alcohol must have been like discovering a bottle of liquid tranquilizer.

After her test of will power had failed (she put the bottles on the table and "just looked at them for a long time, without even touching them"), she poured one of the bottles of scotch into a glass with some ice. At that moment, she probably harbored a strong expectation that the alcohol would provide a source of relief from her tension and anxiety. But at first, nothing happened. "I sipped the drink but didn't feel anything at first, so I poured in the

other one and drank that too." As this was by now her third drink since she broke her abstinence, the AVE was probably of minimal intensity. In addition, her expectation of relief was not immediately satisfied, because of the delay in absorption of alcohol. The conditions were ripe for an overdose: "After that, I drank the bottles of beer in the fridge and then I don't remember what happened."

In terms of the high-risk situations presented in Table 1, I would classify the circumstances leading to the consumption of the first drink as a "social pressure" situation. The components of this situation include the influence of Liane's romanticized image of herself as a drinker and the role that alcohol plays in social situations ("For me, what was going through my mind was that a social drink at parties, lunches, dinners, etc., is condoned and exploited, making me feel extremely self-conscious for not participating"), and the influence of Sharon who acted as a powerful model when she ordered the first drink. When she drank to the point of intoxication, the situation could be classified as falling into the "negative emotional state" category -- attempting to cope with feelings of loneliness and anxiety.

Implications for Treatment and Prevention of Relapse

Many traditional alcoholism programs fail to contend

with the components of the relapse process in their treatment procedures. Often the topic is avoided, as if the very mention of relapse would somehow increase the chances of its occurrence. Instead the emphasis is on the necessity of abstinence as the only "cure" for alcoholism. In my own experience as a clinical psychologist in two inpatient alcoholism programs, the total thrust of the treatment seemed to be to convince the patient that he was indeed an alcoholic, and that he should never drink again. There seemed to be a double message implicit in this approach: you must accept the diagnosis that you are an alcoholic, that you have a disease which is beyond your own personal control; yet the only way to "control" the disease is to desist from drinking on the strength of your own will power or volitional control. From the disease model perspective, there is not much an alcoholic can do once the first drink has been consumed: he is a treatment "fa[illegible] and relapse is inevitable -- drink, drank, drunk.

In recent years, there has been considerable controversy about the possibility of controlled drinking as a treatment goal for some alcoholics (cf. Hamburg, 1975; Lloyd & Salzberg, 1975; Pattison, 1976). The findings of the recent Rand report (Armor, Polich & Stambul, 1976), showing that a sizeable proportion of alcoholics appear to be drinking in

a normal or controlled fashion after discharge from conventional, abstinence-oriented treatment programs, have recently stimulated a barrage of criticism. Programs which have been geared specifically to train alcoholics to engage in moderate or controlled drinking, using newly developed behavioral treatment techniques, have been quite successful (Sobell & Sobell, 1973, 1976). But even in the traditional abstinence-oriented program, there are a number of specific procedures which may reduce the probability of relapse or lessen the likelihood that a single drink will be followed by a bout of intoxication. I will conclude this paper with a brief review of three intervention strategies which appear promising in this regard.

Coping with High-Risk Situations: Specific Alternatives

In a recent treatment study with male alcoholics who participated in an abstinence-oriented program, Chaney and other members of our research team (Chaney, 1976; Chaney, O'Leary & Marlatt, 1977) investigated the effectiveness of a social skill training program designed to help alcoholic inpatients cope with high-risk relapse situations. Using a variety of high-risk situations drawn from our previous research and the self-reports of patients in the program, the therapists conducted a series of skill-training group meetings, in which each patient acted out his responses to

individual situations. Using a training model previously developed by other researchers (e.g., D'Zurilla & Goldfried, 1971; McFall, 1976), Chaney used a combination of techniques, including modeling, therapist coaching, group feedback, behavioral rehearsal, and repeated practice, to teach each patient a variety of specific skills to cope effectively with a full range of high-risk situations. Each subject was assessed on several measures of social skill effectiveness, including standardized tests of assertive behavior and a specially designed Situational Competency Test, administered both prior to and following the training program. In this test, each subject is asked to respond verbally to a high-risk situation presented by a narrator on an audiotape; the subject's response is scored for a number of components related to the quality and overall competency of the reply.

Subjects in the skill training condition were compared with two control groups: a discussion group in which patients talked about their personal feelings associated with the same situations covered in the skill training condition, but did not participate in any of the behavioral techniques; and a third group that received only the usual inpatient treatment procedures. All patients were followed at regular intervals for a period of one year following discharge from the hospital. At the one-year follow-up assessment, the subjects who had received skill training showed a significant decrease

in the duration and severity of relapse episodes compared to the two control groups. In addition, performance on the posttreatment administration of the Situational Competency Test was found to be predictive of subjects' drinking behavior during the follow-up period. Chaney's findings are supported by the results of a laboratory study, in which we found that providing heavy drinkers with a constructive alternative response in a high-risk situation (expression of aggression when provoked to anger in an interpersonal encounter) reduced their subsequent consumption of alcohol in an analogue drinking task (Marlatt, Kosturn & Lang, 1975).

Chaney's study demonstrated that training alcoholics to cope with a variety of standardized high-risk situations is an effective treatment procedure. The results of this program could be enhanced even more by training the alcoholic to be his own behavior therapist. By learning to self-monitor his own urges to drink (by keeping an ongoing daily record of his temptations) the alcoholic should be able to recognize the antecedent conditions leading up to a high-risk situation, and take appropriate preventive action. The client who anticipates these situations in advance should be able to execute self-control procedures to circumvent the problems or minimize their impact. A variety of self-control techniques has been described in the recent

behavioral literature (e.g., Mahoney & Thoresen, 1974; Meichenbaum, 1975). Many of these procedures have been applied to the problem drinker in a recent self-help manual by Miller and Muñoz (1976).

Increased Perception of Control: Global Alternatives

According to the model presented in Figure 3, each time the alcoholic engages in a constructive alternative to drinking in a particular high-risk situation, his perception of personal control should increase in strength. It may also be possible to influence the client's general sense of confidence and control by instructing him or her in a globally effective coping strategy. Techniques such as Jacobson's progressive muscle relaxation (Jacobson, 1938), or a form of meditation, may increase the alcoholic's overall ability to cope with stressful situations. Meditation may be a particularly useful adjunct procedure in the treatment of alcoholism, because it offers an alternative "high" to alcohol. It is also somewhat easier to perform and may be more intrinsically reinforcing than muscle relaxation techniques (cf. Glueck & Stroebel, 1975).

We recently found that if heavy social drinkers take time out each day to perform a simple relaxing exercise, whether it is meditation, muscle relaxation, or simply reading quietly, their daily consumption of alcohol can

be reduced substantially compared to subjects who do not practice such a technique (Marlatt & Marques, 1977). In this study, the regular practice of a relaxation technique led to a significant increase in internal scores on Rotter's (1966) test of locus of control. Thus, subjects who feel more "in control" of their own behavior, as defined by the locus of control measure, show a marked decrement in their day-to-day alcohol consumption. The effects of both skill training (to deal with specific high-risk situations) and a global relaxation technique would seem to be a particularly effective combination in reducing the risk of relapse for alcoholics.

The Programmed Relapse

There is one additional procedure that deserves attention as a possible treatment technique, although it has yet to be evaluated on an empirical basis. One of the strongest factors in the relapse process is the Abstinent Violation Effect described earlier in this paper. Failure to cope adequately with the dissonance and attribution components of this reaction may turn the single slip into a full-blown relapse. It is possible, of course, to prepare the alcoholic to anticipate this reaction during the course of treatment. The therapist could explain the nature of the AVE, and how it may motivate one to continue drinking. The alcoholic in

treatment could be provided with a list of things to remember and to do if a slip does occur. For example, he or she could be given a wallet-sized card to carry around after discharge from the hospital. Such a card might read as follows:

WHAT TO DO IF A SLIP OCCURS

1. Stop after you have finished the first drink. Take half an hour out to consider the following information.
2. A single slip is not all that unusual. It does not mean that you have failed or that you have now lost control over your drinking.
3. You are now probably feeling guilty about what you have done, and blame yourself for having taken the first drink. This feeling is to be expected; it is part of the Abstinence Violation Effect. There is no reason why you have to give in to these feelings and take another drink. The feeling will pass.
4. Look upon the slip as a learning experience. What were the elements of the high-risk situation which led up to the slip? What coping response could you have used to get around this situation? Can you use this coping response now?
5. If you are still having trouble resisting the temptation to drink again, call your therapist. Telephone number:_____. Back-up number:_____.

6. Remember: ONE DRINK DOES NOT MEAN A DRUNK!

Dealing with the pressures of the Abstinence Violation Effect should be a key element in a relapse prevention program. The best way to learn to cope with the demands created by a single slip may be to experience the AVE directly, under supervised conditions. Why not schedule a "programmed relapse" before the alcoholic is discharged from the treatment program, or during a specified follow-up contact period? To experience the effects of a single drink, under a therapist's close supervision, may help the alcoholic to cope with the potential problems of a relapse should it occur in the outside world. A programmed relapse could serve as a "dry run" to iron out difficulties before they occur -- much like a fire drill is used as a prevention measure for future emergencies. The experience may also help the alcoholic in treatment to overcome some traditional beliefs about the magical potency of alcohol. He may come to believe instead that one drink is not strong enough to push him off the wagon.

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Footnotes

1. Throughout this paper, the terms "alcoholism" and "alcoholic" are used in the conventional sense (as in the medical context, which assumes that "alcoholism" is an addiction or a disease state qualitatively different from normal or "social" drinking). Personally, I prefer the terms "problem drinking" and "problem drinker," because of the emphasis on the variety of life problems which may be associated with the excessive use of alcohol. From this perspective, problem drinking lies on one end of a continuum of drinking behavior, with non-problem, light or occasional use of alcohol on the other end. But because the content of this paper deals with the basic assumptions of the traditional "disease" model, I have chosen to use the conventional labels. The same reasons apply to my use of the terms "craving," "loss of control," and "relapse."
2. In the discussion of the case study, names and other identifying material have been changed. The client has given her permission to discuss her case and to quote from her personal notes.
3. I am indebted to one of my students, John L. Rogers, who originally suggested this term.

Table 1

Analysis of Situations in which Relapse Occurred

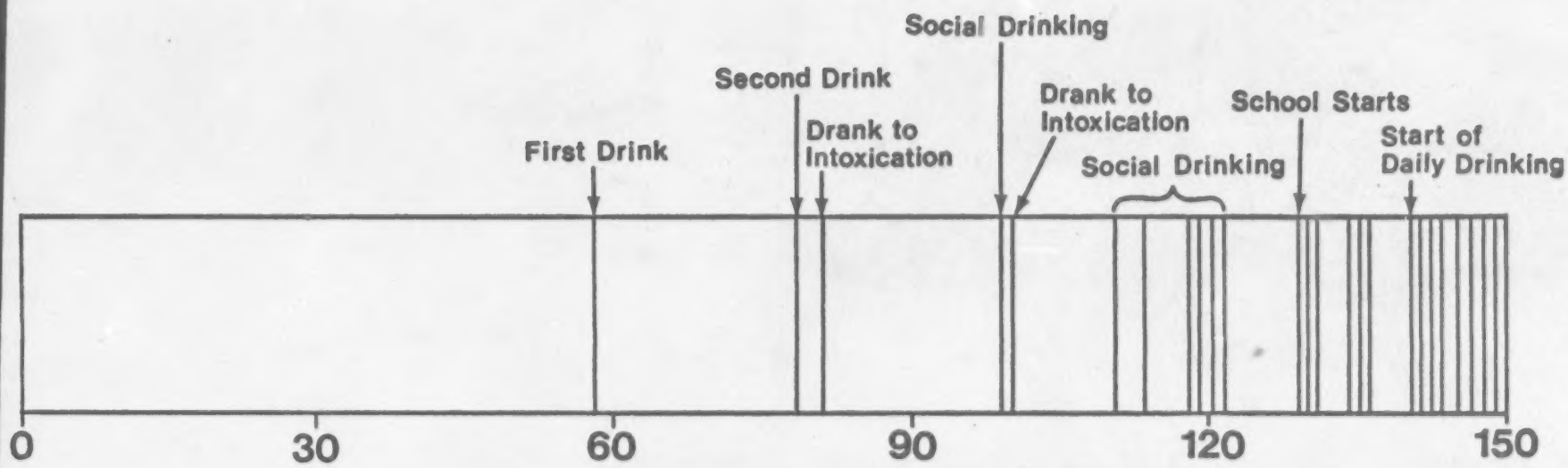
Situation Category	Example	Number	Percent
Frustration and anger	Patient tried to call his wife (they were separated); she hung up on him; he became very angry and took a drink.	14	29%
Social pressure	Patient went with the "boys" to a bar after work. They put pressure on him to "join the crowd" and he was unable to resist.	11	23%
Intrapersonal temptation	Patient walked by a bar, and "just unconsciously walked in, no real reason"; could not resist the temptation to take a drink.	10	21%
Negative emotional state	Patient living alone, no job; complained of feeling bored and useless; could see no reason why he should not take a drink.	5	10%
Miscellaneous other situations	Patient reported that everything was going so well for him that he wanted to celebrate by having a drink.	5	10%
No situation given or unable to remember		3	7%

Figure Captions

Figure 1. Relapse pattern of the client discussed in the case study. Vertical lines refer to drinking episodes during the first 150 days following her discharge from the hospital.

Figure 2. Ludwig and Wikler analysis of relapse.

Figure 3. Cognitive-behavioral analysis of relapse.



Days Following Discharge from Hospital

FIG. 1

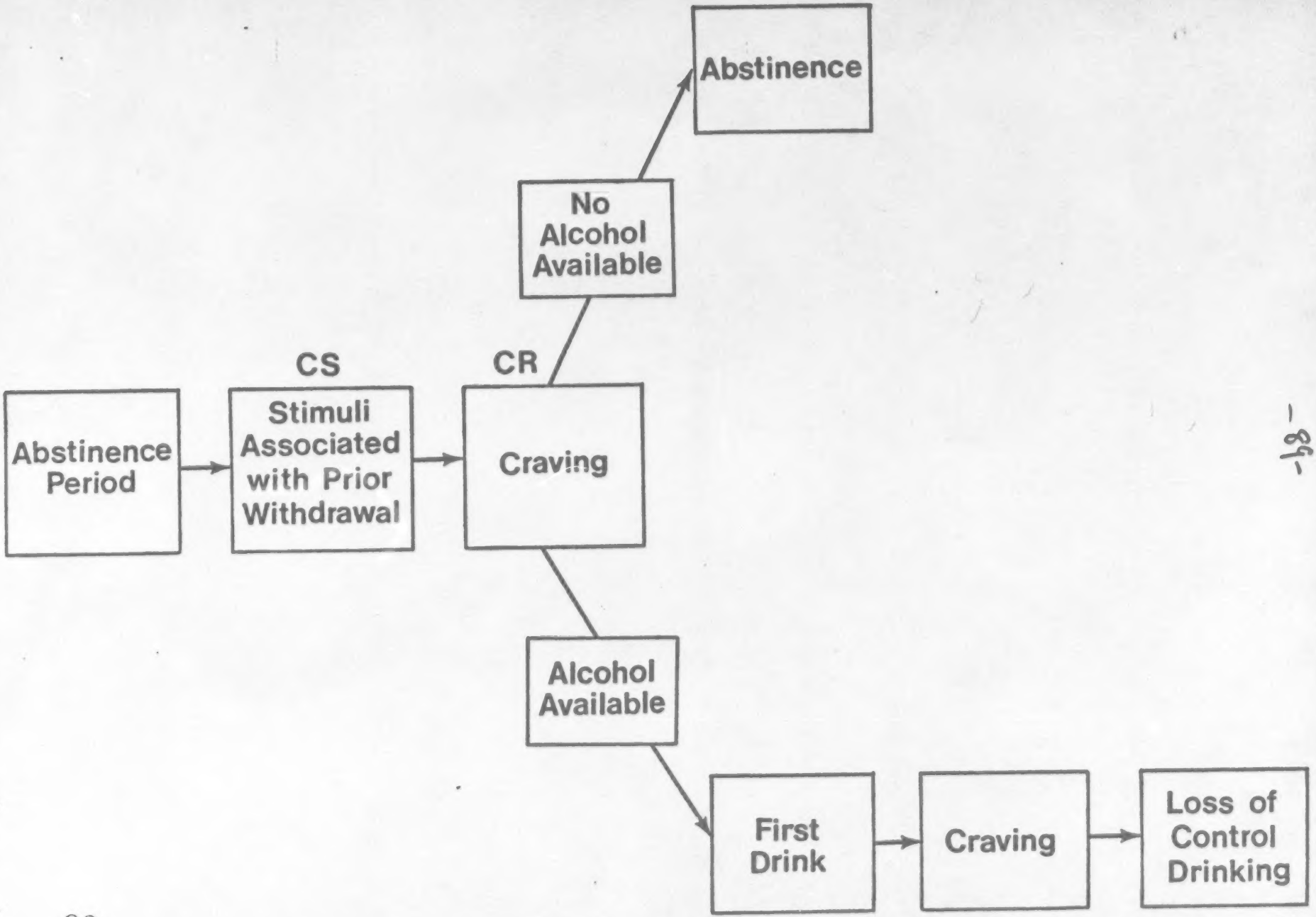


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FIG. 2 Ludwig and Wikler Analysis of Relapse

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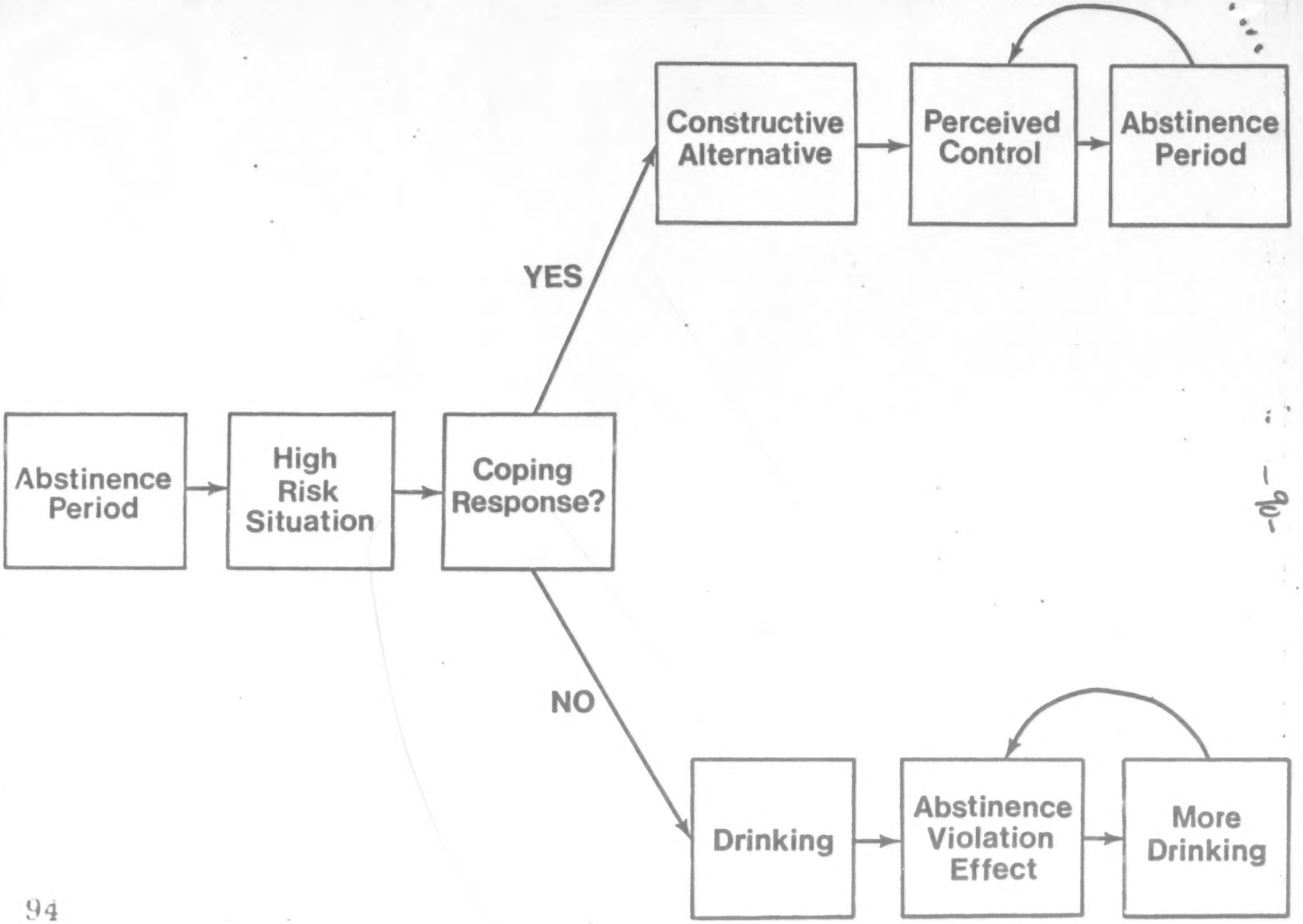

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Fig. 3 Behavior Analysis of Relapse

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